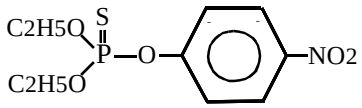
PARATHION

Parathion is a federal hazardous air pollutant and was identified as a toxic air contaminant in April 1993 under AB 2728.

CAS Registry Number: 56-38-2

Molecular Formula: $C_{10}H_{14}NO_5PS$



Parathion is a pale yellow liquid with a faint odor. It is freely soluble in alcohols, esters, ethers, ketones, and aromatic hydrocarbons; and insoluble in water, petroleum ether, kerosene, and spray oils (Merck, 1989).

Physical Properties of Parathion

Synonyms:	phosphorothioic acid; o,o-diethyl o-(4-nitrophenyl) ester; ethyl parathion; DNTP; Alkron; Alleron; Aphamite; Etilon; Folidol; Fosferno; Niran; Paraphos; Rhodiatox; Thiophos
Molecular Weight:	291.27
Boiling Point:	375 ºC
Melting Point:	6 ºC
Density/Specific Gravity:	1.26 at 25/4 ºC (water = 1)
Vapor Pressure:	3.78×10^{-5} mm Hg at 20 ºC
Log Octanol/Water Partition Coefficient:	3.83
Water Solubility:	6.54 mg/l at 24 ºC
Conversion Factor:	1 ppm = 11.91 mg/m^3

(Howard, 1990; HSDB, 1991; Merck, 1989; U.S. EPA, 1994a)

SOURCES AND EMISSIONS

A. Sources

Parathion was registered for use as a pesticide; however as of December 31, 1994, it is no longer registered for pesticidal use in California (DPR, 1996).

B. Emissions

No emissions of parathion from stationary sources in California were reported, based on data obtained from the Air Toxics "Hot Spots" Program (AB 2588) (ARB, 1997b).

C. Natural Occurrence

No information about the natural occurrence of parathion was found in the readily-available literature.

AMBIENT CONCENTRATIONS

At the request of the Department of Pesticide Regulation, the Air Resources Board conducted 24-hour ambient sampling for parathion at several locations in both the San Joaquin Valley (SJV) and Imperial County areas. Sampling was performed in January through February 1986 in the SJV, while sampling in Imperial County occurred in late September through October 1986. Ambient concentrations of parathion ranged from 1 part per trillion (ppt) at a background site to 15.68 ppt at a populated site (ARB, 1986e).

The United States Environmental Protection Agency (U.S. EPA) reported parathion concentrations in Orlando, Florida from 1967-68 that ranged from 10.2 to 25.4 nanograms per cubic meter (ng/m^3) (U.S. EPA, 1993a). Studies of plum orchards showed parathion concentrations near 3,500 ng/m^3 immediately after spraying and 4,100, 394, 149, 21, and 16 ng/m^3 one, two, five, 14, and 21 days after spraying, respectively. Parathion was also detected in the atmospheric fog water from Parlier, Corcoran, and Lodi, California at concentrations of 9,000, 950 and 184,000 nanograms per liter, respectively (Howard, 1990).

INDOOR SOURCES AND CONCENTRATIONS

No information about the indoor sources and concentrations of parathion was found in the readily-available literature.

ATMOSPHERIC PERSISTENCE

When parathion is sprayed on a field or orchard it will exist primarily as an aerosol in the source area and as a combination of vapor and aerosol downwind. The dominant atmospheric loss process for gas-phase parathion is expected to be by reaction with the hydroxyl (OH) radical (Atkinson, 1989, 1994). Based on this reaction, the atmospheric half-life and lifetime of parathion is estimated to be 2.6 hours and 3.8 hours, respectively (Winer and Atkinson, 1990). It is important to note that at solar noon, during the summer months, the half-life for parathion due to reaction with the OH radical is estimated to be less than 1 hour, possibly not inconsistent with observations of the conversion of parathion to paraoxon under field conditions (Woodrow et al., 1978). The atmospheric photooxidation products include paraoxon (Atkinson, 1995).

AB 2588 RISK ASSESSMENT INFORMATION

Parathion emissions are not reported from stationary sources in California under the AB 2588 program. It is also not listed in the California Air Pollution Control Officers Association Air

Toxics “Hot Spots” Program Revised 1992 Risk Assessment Guidelines as having health values (cancer or non-cancer) for use in risk assessments (CAPCOA, 1993).

HEALTH EFFECTS

Probable routes of human exposure to parathion are inhalation, ingestion, and dermal contact.

Non-Cancer: Parathion is a highly potent organophosphate-type cholinesterase inhibitor. Systemic toxicity has resulted from inhalation, ingestion, and dermal exposures. Acute exposure of humans may result in nausea, vomiting, abdominal cramps, diarrhea, excessive salivation, headache, weakness, difficult breathing, blurred vision, convulsions, central nervous system depression, paralysis, coma, and respiratory failure. Depressed red blood cell cholinesterase activity, nausea, and headaches have been observed in chronic inhalation exposure of humans to parathion (U.S. EPA, 1994a). People at special risk are those with a history of glaucoma, and diseases of the cardiovascular, hepatic and renal systems, or central nervous system abnormalities (Sittig, 1991).

The U.S. EPA has not established a Reference Concentration (RfC) for parathion, but has set an oral Reference Dose (RfD) of 0.006 milligrams per kilogram per day based on erythrocyte cholinesterase inhibition in humans. The U.S. EPA estimates that consumption of this dose or less, over a lifetime, would not likely result in the occurrence of non-cancer chronic effects.

No information is available on adverse reproductive effects of parathion in humans. In rats exposed to parathion by injection, a reduced number of offspring and decreased fetal weight were observed, but no malformations were found (U.S. EPA, 1994a).

Cancer: No information is available on the carcinogenic effects of parathion in humans. Limited evidence is available from animal studies. In rats orally exposed to parathion, increased tumors in the adrenal gland in both sexes, and thyroid follicular adenomas and pancreatic islet-cell carcinomas in male rats were observed. The U.S. EPA has classified parathion in Group C: Possible human carcinogen (U.S. EPA, 1994a). The International Agency for Research on Cancer has classified parathion in Group 3: Not classifiable based on no adequate data in humans and insufficient evidence in test animals (IARC, 1987a).